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The Role of Sleep in the Control of Food Intake

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Abstract

Short sleep duration is increasingly recognized as a risk factor for obesity. Sleep is now considered 1 of the 3 lifestyle behaviors, along with diet and exercise, which are closely associated with health. If sleep duration is a causal factor in the etiology of obesity, it must affect energy intake and/or energy expenditure to create a positive energy balance. The preponderance of evidence to date points to an effect of sleep restriction on energy intake that exceeds the added energy costs of maintaining longer wakefulness. Observational studies describe greater intakes of energy, fat, and possibly carbohydrates in short sleepers and this is corroborated by clinical interventions. These intervention studies further provide mechanistic explanations, via alterations in hormonal and neuronal controls of food intake, for an association between short sleep and obesity.

Keywords

sleep duration; energy intake; obesity

Obesity is a major public health problem: In the United States, its prevalence has increased from 13% in the early 1960s to 36% around 2010.[1] Short sleep duration (SSD; typically defined as ≤6 hours) has recently been associated with weight gain and obesity,[2] along with several cardiometabolic risk factors.[3–5] This has led to the dissemination of a report from the Institute of Medicine, focusing on public health issues related to sleep loss,[6] and adequate sleep has been added as a national health priority to Healthy People 2020.[7] *Thus, together with exercise and nutrition, sleep is rapidly being recognized as 1 of the 3 pillars of a healthy lifestyle.*

Alterations in energy expenditure and energy intake have been observed after experimental sleep disruption.[8] However, most studies indicate that the weight gain associated with sleep restriction is likely due to increased energy intake, which surpasses the more modest changes in energy expenditure.[8] Indeed, it has been proposed that the metabolic costs associated with insufficient sleep trigger a set of hormonal, metabolic, and behavioral changes to increase food intake.[9] This article will discuss the impact of sleep disruption on

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energy intake by focusing on (*a*) observational studies of sleep and food intake, (*b*) intervention studies of sleep and food intake, and (*c*) hormonal and cognitive pathways, food intake, and sleep.

Observational Studies of Sleep and Food Intake

Several studies have examined population patterns of food intake relative to sleep. In an analysis of data from the Cleveland Children's Sleep and Health Study, SSD was associated with greater energy intake (~200 kcal), greater proportion of energy from fat, lower proportion from carbohydrates, and greater energy intake in early morning (5:00-7:00 AM).[10] In a study of Japanese factory workers, SSD was associated with irregular meal timing and eating habits, snacking between meals, dining out, lower consumption of vegetables, and greater preference for strongly flavored food.[11] In the United States, dietary records from postmenopausal women revealed that SSD was associated with lower protein intake.[12] An analysis of nationally representative data collected from the 2007-2008 National Health and Nutrition Examination Survey (NHANES) found that self-reported long sleepers (≥9 hours) consumed fewer calories than normal sleepers (7-8 hours).[13] Also, very short (<5 hours), short (5-6 hours), and long sleepers consumed a smaller variety of foods, compared to normal sleepers. In examining nutrient profiles, very short and long sleepers consumed lower protein, carbohydrate, fiber, and fat relative to normal sleepers; long sleepers consumed less sugar; and short sleepers consumed less fiber. These results were observed after adjusting for other dietary intake variables, including total caloric intake.

Some studies have also examined dietary patterns associated with chronotype. For example, a study in German adolescents found that "eveningness" (individuals preferring evening activities and later rising) was associated with increased consumption of fast foods and caffeinated drinks.[14] Sato-Mito and colleagues found that among dietetic students in Japan, later midpoint of sleep (time between wake and bed time) was associated with greater fat, alcohol, noodles, confections, and meat consumption and lower protein and carbohydrate consumption, as well as rice, vegetables, eggs, and milk.[15] Baron and colleagues found that those with later sleep times had shorter sleep duration and later meal times.[16] They also consumed more calories, especially in the evening. In a follow-up study of the same sample, higher caloric intake was associated with carbohydrates consumed after 8:00 PM and amount of both carbohydrates and protein consumed within 4 hours of sleep.[17]

In addition to sleep duration and sleep timing, some studies have examined sleep symptoms and dietary patterns. In a study of middle-aged Japanese workers, difficulty initiating sleep was associated with low protein intake, difficulty maintaining sleep was associated with high protein and low carbohydrate intakes, and overall poor sleep quality was associated with low protein intake.[18] In an analysis of 2007-2008 NHANES data, difficulty maintaining sleep was associated with report of having any special diet, nonrestorative sleep was associated with decreased likelihood of a low fat diet, and daytime sleepiness was associated with endorsement of any special diet but less likelihood of endorsing a special low-fat diet.[19] In addition, difficulty falling asleep was associated with decreased protein and fiber intake; difficulty maintaining sleep was associated with decreased protein, carbohydrate, and fiber intake; nonrestorative sleep was associated with decreased protein and fiber intake; and

daytime sleepiness was associated with decreased protein and fiber and increased sugar intakes.[19]

Thus, population studies demonstrate that SSD is associated with greater energy intake, snacking between meals, and lower protein intake, while poor sleep quality is associated with poor diet quality.

Intervention Studies of Sleep and Food Intake

Laboratory-based studies have attempted to explain the association between SSD and obesity by monitoring energy intake after manipulating sleep. These studies show that sleep restriction compared to a period of habitual sleep duration results in increased energy intake, by approximately 300 to 550 kcal/day, when participants are allowed to freely select their food.[20–25] We also reported that restricting sleep by 4 hours increases 24-hour energy expenditure by ~90 kcal,[26] which would result in a net positive energy balance of >200 kcal/day due to sleep restriction. Positive energy balance as a result of sleep restriction was confirmed by Markwald et al.[27] During short compared to habitual sleep, energy intake was increased by ~6%, whereas energy expenditure was increased by ~5%, resulting in a significant weight gain over the 5-day experimental period. *Taken together, these findings suggest that SSD may cause obesity because of an increase in energy intake that surpasses the added energy costs associated with increased time spent awake.*

In terms of macronutrient composition, an increase in fat intake was described after short versus habitual sleep,[22,25,28] although others reported an increase in carbohydrate intake alone.[24,27] These findings are consistent with the reported increase in appetite for sweet/starchy food after sleep restriction,[29] as well as the observed association between SSD and a high-fat diet.[30] Overall, most laboratory findings to date suggest that sleep restriction increases energy intake, particularly fat and possibly carbohydrate intakes. Not all disruptions in sleep are limited to reduced duration, however, and distinct associations have also been reported for sleep architecture and hunger[31,32] as well as food intake and preference.[33] The proportion of time spent in stage 2 sleep was inversely related to energy intake, and the proportion of time spent in both slow wave sleep and rapid eye movement sleep were inversely related to intakes of fat and carbohydrate.[33] *These findings emphasize the importance of overall sleep quality, in addition to duration, in influencing food choice and energy intake.*

Hormonal and Cognitive Pathways, Food Intake, and Sleep

Leptin and ghrelin are 2 well-studied hormones involved in food intake regulation and may provide a potential hormonal mechanism mediating the SSD–obesity link. Variations in either or both of these hormones could explain the hyperphagia observed after sleep disruption. This is because leptin signals satiety whereas ghrelin triggers hunger and food intake. In parallel to the observed relationship between SSD and greater body mass index, a large cross-sectional study found that SSD was associated with low leptin and high ghrelin levels.[34] An innovative laboratory study measured these hormones while exposing participants to 4-hour or 10-hour time in bed, and observed decreased leptin and increased ghrelin during the short compared to long sleep condition.[29] Subsequent experimental

studies, however, did not show similar results, with some describing either no change in these hormones,[24] or increased leptin[35] and decreased ghrelin,[36] after sleep restriction. Reasons underlying these discrepancies may include differences in energy balance state and degree of sleep restriction within the protocols and a potential modulating effect of sex.[8]

In a study large enough to determine sex differences in the hormonal response to sleep loss, an increase in ghrelin was found in men but not women in response to sleep restriction compared to habitual sleep.[37] In the same study, decreases in glucagon-like peptide 1, a gut hormone signaling satiety, were noted after sleep restriction in women but not in men. *These findings suggest that increased energy intake after sleep restriction may be driven by increased hunger signaling in men and weakened satiety signaling in women.*

It is clear that more work needs to be done to fully delineate the ways in which sleep disruption affects hunger/satiety hormones. While convincing evidence does exist to indicate that alterations in the homeostatic/hormonal control of hunger occur after sleep restriction, the hedonic control of food intake is also likely to be influenced by sleep status.

Two studies demonstrated that neuronal responses to food stimuli differed after periods of restricted versus habitual sleep.[38,39] After a night of total sleep deprivation, activity in the right anterior cingulate cortex in response to viewing food images was increased relative to a night of 8-hour time in bed.[38] Also, after 5 nights of 4-hour sleep opportunity, food stimuli increased activity in the putamen, nucleus accumbens, thalamus, insula, and orbitofrontal cortex, among others, relative to a period of 9-hour sleep opportunity.[39] These brain regions are known to be involved in emotional responses, motivation, and reward systems. Finding that food images trigger greater pleasure and reward centers may be a way by which appetite signals manifest themselves to produce greater incentive or motivation for food consumption.

Interestingly, Killgore et_al[40] showed that activity in the ventral medial prefrontal cortex, a region involved in regulating emotional responses and controlling behavior, in response to viewing food images, was negatively correlated with sleepiness and, in women, also negatively correlated with self-reported overeating. *Thus, it appears that neuronal activity in response to viewing foods is affected by sleep state, which can predispose individuals to overeating.*

Conclusions

It is clear that acute, severe sleep restriction affects energy intake. This has been shown in observational studies and confirmed by intervention studies. Several mechanisms have been postulated to be responsible for this increased drive for food consumption under periods of SSD. There may be a hormonal imbalance between hunger and satiety, although this has been controversial.[8] An increasingly recognized explanation may be enhanced hedonic perceptions of foods resulting from signaling in reward and pleasure centers of the brain, which would drive motivation for food seeking and consumption. Both mechanisms, however, may be at play to account for the increased obesity risk in short sleepers.

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